Remarks

Claims 1 and 3-45 are pending in this application. The Applicants have canceled claim 2. The Examiner withdrew claims 7-20, 25-33, 36-43 and 45 in response to a restriction requirement.

The Examiner objected that claims 1-6, 21-24, 34, 35, and 44 embrace non-elected inventions. The Applicants have amended claims 1-6 by deleting the reference to SEQ ID NOS: 1-4 and 9-126 and replacing it with a reference to SEQ ID NO: 1, only. The Applicants respectfully submit that this satisfactorily addresses the Examiner's objection.

The Examiner rejected claims 1-6, 21-24, 34, 35, and 44 under 35 U.S.C. §§ 101 and 112, first paragraph, stating that the claims are not supported by a substantial utility. The Applicants respectfully disagree.

> The Applicants teach that ABCA5, like the related genes ABCA6, ABCA9, and ABCA10, [is] likely to be involved in the reverse transport of cholesterol, as well as in the membrane transport of lipophilic molecules, in particular, inflammation-mediating substances such as prostaglandins and prostacyclins, or in any pathology whose candidate chromosomal region is situated on chromosome 17, more precisely on the 17q arm and, still more precisely, in the 17q24 locus.

Specification, at ¶ 0027. Accordingly, ABCA5 may be used to treat disease:

> The invention also relates to a pharmaceutical composition intended for the prevention of or treatment of a patient or subject affected by a dysfunction in the reverse transport of cholesterol or in the transport of inflammatory lipophilic substances, wherein the composition comprises a nucleic acid encoding any one of ABCA5, ABCA6, ABCA9, and ABCA10 proteins. . . .

Specification, at ¶ 94. The Applicants provide several examples of relevant diseases:

> Various diseases linked to HDL deficiency have been described, including Tangiers disease, FHD disease, and LCAT deficiency. In addition, HDL-cholesterol deficiencies have been observed in patients suffering from malaria and diabetes. The deficiency involved in Tangier and/or FHD disease is linked to a cellular defect in the translocation of cellular cholesterol that causes a degradation of HDLs and leads to a disruption in lipoprotein metabolism.

Specification, at ¶ 0019 (citations omitted). That ABCA5 is associated with such diseases is no surprise, because

> Several ABC transport proteins that have been identified in humans are associated with diseases. For example, cystic fibrosis is caused by mutations in the CFTR (cystic fibrosis transmembrane conductance regulator) gene. Moreover, some multiple drug resistance phenotypes in tumor cells have been associated with the gene encoding the

> MDR (multi-drug resistance) protein, which also has an ABC transporter structure. Other ABC transporters have been associated with neuronal and tumor conditions (U.S. Pat. No. 5,858,719) or potentially involved in diseases caused by impairment of the homeostasis of metals. Likewise, another ABC transporter, designated PFIC2, appears to be involved in a progressive familial intrahepatic cholestasia form, this protein potentially being responsible, in humans, for the export of bile salts.

Specification, at ¶ 0006.

In view of the foregoing, the Applicants respectfully submit that ABCA5 has a substantial utility – the treatment of the various diseases recited in the specification – and respectfully request that the Examiner withdraw the rejection under §§ 101 and 112, first paragraph.

The Examiner rejected claims 1-6, 21-24, 34, 35, and 44 under § 112, first paragraph, stating that the specification describes only SEQ ID NO: 1 but not sequences that are at least 80% similar to it or that would hybridize to it under high stringency conditions. The Applicants respectfully submit that one of ordinary skill in the art would readily know how to make such sequences, especially in view of the Applicants' disclosure. For example, the Applicants provide detailed hybridization protocol at ¶¶ 0221-0245, descriptions of fragments at ¶ 0262, variants at ¶¶ 0264-266, and 0270-276, mutations at ¶ 0278, and provides specific examples of the amino acid substitutions, deletions, and additions possible. The Applicants therefore respectfully request that the Examiner withdraw the rejection of these claims under

§ 112, first paragraph

The Examiner objected to claims 1-6, 21-24, 34, 35, and 44 under 35 U.S.C. § 102(e) as being anticipated by the Hu reference. The reference appears to show, at most, 85.1% identity with SEQ ID NO:1, and therefore cannot anticipate claims directed to that sequence. As to other claims, the Applicants have amended them to require at least 90% identity with SEQ ID NO:1. For these reasons, the Applicants respectfully request that the Examiner withdraw the objection under § 101(e).

The Applicants respectfully submit that the claims, as amended, are in condition for allowance, and respectfully request early, favorable action on the application. Should the Examiner believe that an interview would advance the prosecution of this application, the Applicants invite her to contact the undersigned at 908.231.3444.

Respectfully submitted,

Joel B. German, Reg. No., 48,676
Attorney/Agent for Applicant

Aventis Pharmaceuticals Inc.
Patent Department
Route #202-206 / P.O. Box 6800
Bridgewater, NJ 08807-0800
Telephone (908) 231-3444
Telefax (908) 231-2626

Aventis Docket No. ST00034 US NP